RESEARCH ARTICLE

Exercise causes oxygen desaturation and hypercapnia in stable chronic obstructive pulmonary disease patients

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ABSTRACT

Background: Assessment of severity of chronic obstructive pulmonary disease (COPD) is the cornerstone of therapy. Spirometric measurements have traditionally remained as the popular diagnostic tool of choice. Oxygenation and carbon dioxide removal cannot be assessed by Spirometry alone, especially during exercise. Therefore, we studied whether desaturation and hypercapnea occur in response to exercise in COPD patients. Aims and Objectives: To know whether bicycle pedaling as an exercise can unravel the gas exchange abnormalities and airflow limitation that might be precipitated by physical activity. This is done by estimating the changes in Oxygen saturation by pulse oximetry, and by estimating the changes in Blood CO2 levels by capnography. Materials and Methods: Thirty stable COPD patients and controls were included for the study. Here we measured the change in oxygen saturation from rest to submaximal exercise (done using bicycle ergometry). Concomitantly, we measured the change in carbon dioxide levels of expired air from rest to submaximal exercise. Results: We found that COPD patients experience oxygen desaturation. ΔSaO2 (difference between resting and exercise SaO2) was only 1% in controls whereas 8.86% in COPD. Hypercapnia occurred in response to a submaximal exercise in COPD patients (End tidal carbon dioxide of 48.87 mmHg). We also found that they become tachypneic and show greater degree of exhaustion. Conclusion: Our study points out that exercise-induced desaturation and hypercapnia are a definite occurrence in COPD patients. It is a marker of progressive disease. It can be used as a form of stress test for the pulmonary system.

KEYWORDS: Chronic Obstructive Pulmonary Disease; Exercise Induced Desaturation; Exercise Induced Hypercapnia; Tachypnea

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic respiratory disorder that progresses slowly and is characterized by an obstructive ventilatory pattern, which is rarely reversible, very often related to tobacco smoking and which can lead to chronic respiratory failure. Bronchial obstruction is defined in relation to a drop in forced expiratory volume in 1 s (FEV1)/forced vital capacity ratio ≤70%.[1] COPD is one of the commonest lung diseases in India. COPD and other respiratory diseases are the second (10.2%) leading cause of death in the population aged 25–69 years in India.[2] Cardiac stress to assess the sufficiency of coronary blood flow is a well-known clinical diagnostic tool. It is done by assessing the ischemic electrical changes in response to metabolic stress. In this case, usually induced by making subject to walk or run on a treadmill.

Stable COPD patients may have absent or mildly compromised oxygenation at rest, which may become more apparent during exercise. COPD patients may be normocapnic at rest but may exhibit hypercapnia during
exercise. Therefore to know whether COPD patients exhibit the exercise-induced changes in oxygenation and expired air CO₂ levels, this study was undertaken. This study aimed to know whether bicycle pedaling as an exercise can unravel the gas exchange abnormalities and air flow limitation that might be precipitated by physical activity. Some studies have used six-minute-walk test to assess exercise-induced desaturation. The procedure can assess the changes in SaO₂ and end tidal carbon dioxide (EtCO₂) only at the completion of the walk test. This study assesses continuous changes in SaO₂ and EtCO₂ even as the subject is performing the exercise. This gives a better understanding about the intra-exercise changes in oxygen and carbon dioxide status.

MATERIALS AND METHODS

Ethical clearance was obtained from the Institutional committee. The Ethical committee registration number is ECR/486/Inst/KA/2013/RR-16 and our study ethical clearance letter number is KIMS: ETHCS COMM: 675: 2021-22 the meeting for which was held on November 15, 2018, and November 16, 2018. Informed consent was taken. Thirty known cases of COPD and 30 controls were included in the study. The subjects were aged between 25 and 60 years. They had COPD for at least 5 years. Patients who were on treatment like bronchodilators and steroid inhalers were also included in the study. Healthy individuals without obvious pulmonary disease were taken as controls. Subjects with a history of status asthmaticus, hypertension, congestive cardiac failure, ischemic heart disease were excluded from the study.

Vital data and anthropometric measurements were taken for subjects and controls. They were made to sit on the bicycle ergometer for 5 min. Resting values of heart rate, respiratory rate, SpO₂ and EtCO₂ were recorded. Submaximal exercise was defined as the level of exercise at which the subject reached 80% of the predicted maximal heart rate. Predicted maximal heart rate was calculated using the formula: 220 – age.[11] Subjects were asked to pedal on the bicycle with increasing speed until they reached the target heart rate. They were instructed to maintain that speed for at least 2 min. Heart rate, respiratory rate, SpO₂ and EtCO₂ were recorded again during exercise. The change from resting value was noted.

Statistical Analysis

The statistical analysis was done using student’s t-test. Sample size was calculated using Cochran Formula. We took a sample size of 30 as round figure to get better statistical results. P < 0.05 was taken as significant.

Instruments and Devices Used

Multiparameter monitor model VISMO 10 manufactured by Nihon Kohden Japan, the monitor was used to record SpO₂, EtCO₂, Pulse rate, non-invasive blood pressure and respiratory rate. Bicycle ergometer, model Fit King S-257 displaying speed, calories, and distance was used to administer submaximal exercise. USB Spirometer model Easy on PC manufactured by NDD Medzteknik Switzerland. This was used to assess the lung function parameters of COPD patients.

RESULTS

A total of thirty known cases with COPD and 30 controls were enrolled in the study. The mean age for cases and controls were 48.7 ± 10.2 years and 36.2 ± 9.2 years, respectively. The mean weight and height of cases were 59.1 ± 5.12 kg and 152.6 ± 3.96 cm, respectively. The mean weight and height of controls were 70.44 ± 6.01 kg and 156.73 ± 9.26 cm respectively. The difference in height between cases and controls was significant, also controls had greater mean body weight than cases. The resting heart rate and blood pressure for controls were 85.7 ± 13.3 bpm and 116/77 mmHg, respectively. The resting heart rate and blood pressure for cases were 85.4 ± 6.03 bpm and 120/79 mmHg, respectively. The target heart rate was achieved among cases and controls during submaximal exercise. Blood pressure increased to 146/96 mmHg in controls and 150/88 mmHg in cases. Cases were older than controls. Cases were leaner than their normal counterparts. The lower body mass in cases could be attributable to the inflammatory process in COPD leading to cachexia and also skeletal muscle mass loss, also greater loss of calories due to greater workload for respiratory muscles in COPD. The resting heart rate and blood pressure were similar in both groups.

Delta SpO₂ is the drop in oxygen saturation of blood when a subject performs exercise. It is the difference between resting SpO₂ and exercise SpO₂. COPD patients in our study exhibited marked degree of exercise-induced oxygen desaturation. Exercise-induced desaturation (EID) is defined by two criteria. A drop in SpO₂ of >4% or a nadir of 88%,[4-7] COPD patients exhibited a drop of more than 8% after exercise, but healthy controls showed only a drop of 1%. We also noted that in some healthy controls, exercise actually produced a rise of SpO₂. The data are shown in Table 1.

Delta EtCO₂ is the rise of expired air CO₂ levels when the subject performs exercise. It is the difference between resting EtCO₂ and exercise EtCO₂. This study revealed that COPD patients suffer from hypercapnia during exercise and therefore retain greater amount of CO₂ during exercise. We noted that controls never crossed the EtCO₂ value beyond 45mmHg. 45 mmHg of EtCO₂ is the cut-off value to indicate Hypercapnia.[8,9] Also, few healthy controls actually showed reduction in EtCO₂ at peak exercise. The data is shown in Table 1.
The resting respiratory rate in controls and cases was 16.46 ± 3.22 and 20.46 ± 4.94 per minute respectively. Exercise respiratory rate in controls and cases was 28.6 ± 5.6 and 34.4 ± 6.6 per min respectively. COPD cases had slightly greater respiratory rate at rest which may indicate that they might be in dyspnea even at rest. COPD cases showed a greater rise in respiratory rate at exercise which may show obvious inefficiency of respiratory muscle effort.

DISCUSSION

The main findings of this study are significant desaturation that occurs during exercise in COPD. Delta SaO₂ is the difference between resting SaO₂ and Exercise SaO₂. This will tell how much reduction has occurred in pulmonary reserve due to COPD. Another important finding of this study is the occurrence of hypercapnia during exercise in COPD patients. This may possibly point towards multiple pathophysiologic mechanisms like expiratory flow limitation, diffusion defect, and reduction in skeletal muscle (respiratory muscle) strength. These may together contribute to inadequate wash out of carbon dioxide during exercise, whose production increases markedly during exercise.

Patients with COPD are known to have varying degrees of hypoxemia. Some are normoxic at rest and begin to desaturate during exercise. Van Gestel et al. (2012) performed a study to find the associations between FEV₁, DLCO, and resting SaO₂. They found the best predictor of EID was FEV₁. They measured FEV₁, DLCO, SpO₂ at rest and during a 6-min walking test. A drop in SpO₂ of >4% or a nadir upto <90% was defined as EID. They further quote that desaturation during exercise is associated with higher mortality. They performed a 6 min walk test and measured SpO₂.[14]

Patients with COPD are assessed of their disease severity by conducting many tests including diffusing capacity of the lungs for carbon monoxide (DLCO), pulmonary artery (PA):A ratio (PA diameter to aorta diameter ratio as an index of pulmonary hypertension), area of emphysema on computed tomography (percentage of low attenuation area). Fujimoto et al. (2017) conducted a study where they correlated the above parameters with desaturation–distance ratio (DDR). DDR is an index derived from the distance traveled and level of desaturation during a six-minute walk test. They collected the data in 74 stable COPD patients. Relationships between DDR and other parameters were analyzed with Spearman’s rank-correlation analysis. They found out that DDR correlated significantly with FEV₁, DLCO and PA: A ratio. DDR was highly accurate in predicting severe airway obstruction, defective diffusing capacity, severity of emphysema, and enlargement of PA.[10]

Behnia et al. (2017) assessed lung diffusion capacity and exercise capacity in COPD patients. They measured hemoglobin, spirometry, single breath DLCO, Intra Breath DLCO, and pulmonary blood flow and followed by progressive cycle ergometry to exhaustion with measures of oxygen saturation (SaO₂) and expired gases. They found SaO₂ (%) 96 ± 2 at rest, 95 ± 3 at first workload and 94 ± 3 at peak exercise. They also found PetCO₂ (mmHg) 35 ± 5 at rest, 37 ± 4 at first workload, and 37 ± 5 at peak exercise. These findings are in close confirmation with our study. They further opine that ratio of single breath DLCO to pulmonary blood flow appears to be a better predictor of exercise capacity than more classic measures such as exercise-induced desaturation and exercise-induced hypercapnia.[11]

In this study, we found that most COPD patients exhibited normocapnia at rest but hypercapnia during submaximal exercise. This makes us to propose that exercise-induced hypercapnia can be used as objective indicator of the severity of the disease process. In an article by Poon et al. (2015), they describe that late-stage COPD patients are prone to CO₂ retention. They attribute this to ventilation-perfusion mismatch. However, patients with mild or moderate COPD too suffer similar ventilatory inadequacy but they maintain normocapnic at rest, and during exercise with an increased respiratory effort to overcome the wasted dead space ventilation. In severe COPD, exercise-induced hyperventilation progressively becomes ineffective as the disease progresses, causing hypercapnia at peak exercise, this could be due to ventilatory limitation due to increasing expiratory flow limitation and dynamic lung hyperinflation.[12]
arterial oxygen desaturation and with concomitant increases in dynamic end-expiratory lung volume/total lung capacity (EELV/TLC). The subgroup of patients who retained CO\(_2\) in response to exercise demonstrated greater ventilation-perfusion inequalities and greater mechanical constraints on ventilation than the subgroup of patients who did not retain CO\(_2\). In our study none of patients were non-retainers of CO\(_2\) during exercise, all patients showed EID too. We, therefore, propose that Exercise hypercapnia in COPD is good measure of deranged ventilation-perfusion ratio and dynamic EELV/TLC.\(^{[13]}\)

**CONCLUSION**

This study examined the effect of exercise on oxygen saturation and expired carbon dioxide levels in COPD patients. We found that submaximal exercise done using bicycle ergometry produces hypoxemia or oxygen desaturation in COPD patients who display normal oxygen saturation at rest. COPD patients also displayed hypercapnia in response to submaximal bicycle exercise even if they are normocapnic at rest. So we opine that submaximal exercise can unravel the hidden gas exchange abnormalities that exist in COPD patients.

**REFERENCES**


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